Conclusion: Familial patients with spontaneous cervical carotid artery dissection are at high risk for recurrent or multiple cervical carotid artery dissections.

Summary: Only a minority of patients with spontaneous carotid artery dissection have a clinical diagnosis of a connective tissue disorder. However, dermatologic biopsy specimens in young patients with spontaneous carotid artery dissection have shown abnormalities in up to 50% to 60%, despite the absence of clinical signs of connective tissue disorder (Neurology 2001; 57:24-30).

The authors identified seven families with 15 patients with spontaneous carotid artery dissection. Patients were examined by a dermatologist, a neuroradiologist, and a neurologist for clinical characteristics of an underlying connective tissue disorder. Eleven patients had a skin biopsy. Coding sequences COL3A1, COL5A1, COL5A2, and part of COL1A1 were also examined.

The mean age at the time of the first spontaneous carotid artery dissection was 36.2 years (median, 32 years; range, 18 to 59 years). There were nine men and six women, and two patients had bilateral spontaneous carotid dissection. One patient had five dissections within an 8-year period. There was a high correlation among families between affected vessels (ie, vertebral or carotid arteries, or both) and between age at the first dissection. All but one patient analyzed with skin biopsies showed normal connective tissue morphology. One patient had mild findings consistent with vascular Ehlers-Danlos syndrome type 4. Four patients were identified with mutations in COL genes.

Comment: Patients with familial spontaneous carotid artery dissection are younger than the general cohort of patients with spontaneous carotid artery dissection. Microscopic analysis of skin biopsy specimens is essentially unrevealing. Younger patients with spontaneous carotid artery dissection should perhaps be counseled about a familial tendency for this disease, but it does not appear that genetic testing, at least as we currently understand it, is helpful.

Long-term survival in patients presenting with type B acute aortic dissection: Insights from the International Registry of Acute Aortic Dissection

Tsai TT, Fattori R, Trimarchi S, et al; and the International Registry of Acute Aortic Dissection (IRAD). Circulation 2006;114:2226-31.

Conclusion: There is a high mortality rate in patients with acute type B aortic dissection who survive to hospital discharge, with almost one in four patients dead at 3 years.

Summary: Patients with acute type B aortic dissection generally do well in the hospital, with 90% surviving to hospital discharge with effective antihypertensive therapy (JAMA 2000;283:897-903). Late follow-up of such patients has not been well studied and has been primarily restricted to single-center reports. The authors used data from a contemporary registry of acute type B aortic dissections to analyze long-term survival in such patients.

Data were derived from the International Registry of Acute Aortic Dissection (IRAD). Patients were accumulated from 1996 to 2003. The basis of this study was 242 consecutive patients discharged from the hospital alive with acute type B aortic dissection. Survival was determined with Kaplan-Meier survival curves. Cox proportional hazards analysis was used to identify predictors of mortality during follow-up.

Three-year survival for patients treated medically was 77.6% ± 6.1%, 82.8% ± 18.9% for those treated surgically, and 76.2% ± 25.2% (log rank $P = .61$) for those treated with endovascular therapy. Predictors of mortality during follow-up included a history of aortic aneurysm (hazard ratio [HR], 2.17; 95% confidence interval [CI], 1.03 to 4.59; $P = .04$), history of atherosclerosis (HR 2.41; 95% CI 1.32 to 4.66; $P < .01$), plural effusion (HR, 2.56; 95% CI, 1.18 to 5.58; $P = .02$), hypotension and shock in the hospital (HR, 12.5; 95% CI, 3.21 to 48.21; $P < .01$), and female gender (HR, 1.99; 95% CI, 1.07 to 3.71).

Comment: The predictors of mortality after acute type B aortic dissection identified in this report have all been previously identified. No differences in survival existed between medically managed, surgically managed, and endovascularly managed patients. This was not a randomized study, however, and it is possible that the patients treated with either surgical or endovascular techniques had additional risk factors for long-term mortality. The bulk of the data, however, do suggest that it is going to be difficult to acquire sufficient patients with sufficient power to test the hypothesis of whether immediate endovascular therapy of acute type B dissection provides better long-term results than standard medical management and intervention-only for complications of dissection or failure of medical management.

Sonographic elasticity imaging of acute and chronic deep venous thrombosis in humans

Rubin JM, Xie H, Kim K, et al. J Ultrasound Med 2006;25:1179-86.

Conclusion: Ultrasound-based elasticity imaging may help distinguish chronic from acute thrombosis and thus be useful in the clinical evaluation of patients with post-thrombotic syndrome.

Summary: Distinguishing acute from chronic venous thrombosis is difficult with ultrasound B-mode imaging alone but may be important in a patient with a history of venous thrombosis and new symptoms in the leg. Under such circumstances, determining the age of the thrombus has significant clinical implications. One method of potentially aging venous thrombus is sonographic elasticity imaging. This technique allows direct assessment of tissue hardness. Because deep vein thrombi harden as they age, the technique may be possible to use in distinguishing acute from chronic venous thrombus.

The authors of this study analyzed data in 46 patients: 23 had acute venous thrombi (mean age, 5.7 days) and 23 had chronic venous thrombi (>8 months). A 5-MHz linear array transducer was used to subject each thrombus to free-hand external deformation. Strains in the thrombi were normalized to average strain between the skin surface and the back wall of the vein. Thrombus echogenicity was measuring by comparing the adjacent arterial lumen with the echogenicity of the thrombus.

The acute cases had a median normalized strain magnitude of 2.75 (interquartile range, 2.4 to 3.71). In chronic cases, the mean normalized strain magnitude was 0.94 (interquartile range, 0.48 1.36, $P < 10^{-7}$). Echogenicity differences between the populations were also highly significant ($P < 10^{-5}$).

Comment: Reliably determining the age of a venous thrombus is one of the Holy Grails of noninvasive vascular testing. The population studied here, however, was somewhat artificial compared with the clinical situation. The current population consisted of patients with known acute vs known chronic thrombi. Sonographic elastic imaging needs to be evaluated in patients with a combination of acute and chronic venous thrombi to more mimic the clinical question of determining thrombus age in a patient with acute symptoms and known previous venous thrombosis.

Hypertension accelerated experimental abdominal aortic aneurysm through upregulation of nuclear factor κB and Ets

Shiraya S, Miwa K, Aoki M, et al. Hypertension 2006;48:628-36.

Conclusion: Hypertension accelerates progression of experimental abdominal aortic aneurysms (AAAs) by upregulation of Ets and nuclear factor κB (NF κB).

Summary: The pathogenesis of AAA is complex but appears to involve upregulation of certain matrix metalloproteinases (MMPs). Experimental AAAs are characterized by upregulation of transcription factors, NF κB, which regulate transcription, and Ets, which regulates expression of MMPs. The role of hypertension in pathogenesis of AAA is controversial, with some studies suggesting it contributes to aneurysm formation and other studies suggesting it is not an independent risk factor for aneurysm formation.

In this study, the authors used an experimental model of AAA to investigate the effects of hypertension on transcription factors NF κB and Ets. They also investigated the effects of hypertension on progression of AAA. Experimental AAAs were produced by elastase perfusion in both normotensive and hypertensive rats. Compared with normotensive rats, the size of the AAAs increased more rapidly in the hypertensive rats. Western blot analysis indicated the expression of MMP-2, MMP-3, MMP-9, and MMP-12, as well as that of intercellular adhesion molecule, was increased in hypertensive rats. This was accompanied by upregulation of NFκB and Ets. There was also increased activity of MMPs in the aortas of hypertensive rats vs those of normotensive rats.

Chimeric decoy oligodeoxynucleotide (ODN) inhibits expression of NFκB and Ets. Transfection of the ODN resulted in significant inhibition of aortic dilatation in both hypertensive and normotensive rats. Transfection with chimeric decoy ODN resulted in significant inhibition of degeneration of elastic fibers in aortas of both normotensive and hypertensive rats. Expression of MMP-2, MMP-3, MMP-9, and MMP-12 and intercellular adhesion molecule was decreased by chimeric decoy ODN. This was accompanied by inhibition of macrophage migration.

Comment: The study indicates hypertension accelerates progression of AAA in an experimental rat model. Perhaps inhibition of NF κB and Ets may someday be used as a method of decreasing aneurysm expansion in both normotensive and hypertensive patients.